

Introduction

A parasite, by definition, is an organism that benefits while the host is harmed in the process. Viruses, or virions, use host cell machinery to achieve replication and infection at the expense of the host (Cann 2008). Therefore, viruses are considered as parasitic entities. Viruses are non-living ubiquitous microorganisms. Living cells are characterized by features like cellular reproduction, cellular homeostasis, energy metabolism, and external responsiveness whereas viruses lack these characteristics on their own. However once inside of a host, viruses can perform the features of living cells like genetic replication and generate progeny virions. As a result, there is a debate over whether viruses are alive or not. Since viruses are dependent and cannot self-sustain on their own but exhibit some features of life in certain conditions, viruses are generally considered as non-living organisms.

Viruses are everywhere, infecting various species ranging from plants, vertebrates, invertebrates, bacteria, fungi, and even algae (Strauss and Strauss 2008). Viruses vary in size ranging from 16 nm to more than 300 nm in diameter (Modrow et al. 2013). Viruses are very diverse in classification and type (López-García and Moreira 2012). The nucleic acids can differ between viruses and take on different shapes. To protect the genomic material, viruses have a layer called the capsid to help retain infectivity outside of the host cell (Roos et al. 2007). In addition to this protective layer, a lipid layer may or may not be present surrounding the capsid. Viruses with and without this layer are referred to as enveloped and naked respectively. Viruses are simply nucleic acids and proteins. Replication of progeny virions cannot occur without the nucleic acid. Viral structures, like protein spikes, assist in attachment and uptake into the host. A general sequence of events that viruses proceed through once inside a host cell is as follows: attachment, entry, uncoating, replication, assembly, maturation, and release. Attachment and entry are the binding of the viral antigen with the host receptor. This is like a key and a lock analogy. A specific key, the viral antigen, will fit into a specific lock, the receptor on the host cell, to open. Once inside the cytoplasm, the virus needs to un-coat to release the materials needed for replication. This is where progeny viral nucleic acids and proteins are produced. Assembly refers to the capsid and nucleic acids, along with other viral proteins needed, to make a complete virion. The process of maturation is complete when the virus is released from the host cell and is able to infect other cells.

Viruses have been present throughout the history of life. Viruses are quasispecies that co-exist with other organisms in all aspects of life (Villarreal 2008). Quasispecies refers to a population with a large pool of possible genomes. Genetic diversity is necessary for natural selection to act on a population to affect fitness. Viruses exist as genetically diverse entities due to the rapid accumulation of mutations resulting in various fitness. Viral interactions with compatible hosts are essential for the existence of viruses which likely co-evolved in diverse lineages of life. Endogenous viral elements, which are viral nucleic acids found within non-viral species, are evidence of animal-virus relationships. In Koala bears with leukemia, endogenous viral elements from retroviruses, enable better survivability. In humans, the major histocompatibility complexes (MHC) present foreign particles on cell surfaces like retroviral proteins. This may provide insight into the origins of the adaptive immune system since viral antigens were not recognized as self. Exposure to diseases and viruses acted as a selective pressure in early human evolution (Van Blerkom 2003). Despite dangerous consequences of viral infections, viruses have contributed to genetic polymorphism, which are differences in the DNA sequence among alleles. Such polymorphisms in the human MHC can strengthen the

immune response. The presence of viral DNA in the genomes of various species support the ancient origins of viruses.

Around the late 1890s the structure and identity of a virus was first classified after a virus was isolated from tobacco plants (Oldstone 2014). The discovery of the tobacco plant virus by Ivanovsky and Beijerinck followed the work of Pasteur. Pasteur isolated and cultured bacteria through filters, and viruses were isolated with a similar methodology. However, the unknown infectious particles still remained at the bottom of the filtering apparatus. The discovery of a virus was proven when the invisible filtrate at the bottom of the filtering apparatus was used to inoculate a healthy plant that became diseased. The pore size of the filtration device is small enough to trap bacteria but not viral particles. The size of viral particles is smaller than bacteria. As such, electron microscopes, which were not available until the 1930s, were required to examine the extreme miniature sizes of viruses. Since the early studies in microbiology, viral identification has allowed for advancement in biotechnology and pharmaceutical interventions.

Viruses exist where there are living organisms. A virus without a host is rendered inactive, for viruses have no mechanisms to reproduce outside of a host cell. As such, viruses can exist in terrestrial or aquatic environments due to numerous living organisms as potential host (Kimura et al. 2008). Viruses that infect bacteria are called bacteriophages, or phages for short. The ecological impact, and possible benefits, of viruses is inevitable between these microbes. For example, bacteriophages influence biogeochemical cycles via their interactions with soil bacteria (López-García, and Moreira 2012). This can be beneficial in controlling microflora biomass and competition among bacterial species.

The term virus has a negative connotation among the general population. Viruses are commonly associated with infectious diseases that can cause human illness and in extreme cases, death. For example, influenza virus, parvovirus, or herpes viruses can cause symptoms like fever, diarrhea, and sores respectively in infected individuals. Other viruses, like Ebola, exhibit severe virulence. Social media networks and news broadcast are the major sources of information to reach wide audiences among the population. This can be problematic as false information and misleading beliefs are disseminated among the viewers and listeners. This “infodemic” is especially an issue in locations where the population varies in level of media literacy and education (Melki et al. 2021). Those who use news broadcast and social media as the primary source of attaining information are more likely to believe in false information compared to educated individuals following the guidelines of the government and health professionals. Media literacy is essential for preventive measures and mitigation of a pandemic. The misleading infodemic contributes to the widespread and prolonged COVID-19 pandemic worldwide.

Despite unfavorable attitudes and thoughts surrounding viruses, viruses can have applications in future medicinal usage through virotherapy (Mietzsch and Agbandje 2017). These can include virus cancer therapy, genetic therapy, and vaccine therapy. In addition, vaccine delivery by virosome particles is also possible (Saroja et al. 2011). The purpose of a vaccination is to stimulate a pseudo infection and prepare the immune system to respond quickly when exposed to the actual virus. A factor in vaccine effectiveness is particle size when administered due to its effect on cell uptake. The virosome is a lipid membrane resembling an empty shell of the influenza virus, like an empty procapsid, that does not contain the genome. The antigens, or protein markers, of the influenza virus are retained on the surface of virosome. This structure facilitates host-receptor interactions, and ultimately vaccine delivery by endocytosis into the target cell. An immunity response will then ensue, which is beyond the scope of this review. Novel medical innovations and treatments are necessary in a world of infectious diseases.

Rhinovirus, the common cold, and influenza virus, the flu, infections are frequent year-round (Visseaux et al. 2017). Other factors of the year, like seasonal changes, also influence infectivity rate, where the winter seasons tend to have higher exposure.

Viral pathogens are generally confined to a particular set of closely-related host species (Slingenbergh et al. 2004). Animal viruses only infect animals, and plant viruses only infect plants. However, viruses specific to certain animal species can sometimes cross into humans, a process known as zoonosis. Zoonosis is attributed to agriculture and urbanization where exposure to livestock and wildlife increase the chances of viral host jump. The COVID-19 pandemic originated in the city of Wuhan, China where the virus, associated with and found in bat species, crossed barriers and infected humans (Mackenzie and Smith 2020). How the coronavirus crossed over to humans, however, is unclear.

Coronaviruses are categorized within the family Coronaviridae. The prefix of coronavirus means a crown or a circle with rays, like the sun, which resembles the appearance of the coronavirus. Coronavirus is abbreviated as COVID or SARS-CoV. These abbreviations stand for coronavirus disease and severe acute respiratory syndrome-coronavirus respectively. The numbers that follow the abbreviation correspond to the year of the outbreak. For example, COVID-19 is the outbreak of the year 2019. SARS-CoV-2, or COVID-19, is the second outbreak where SARS-CoV-1 occurred in 2003. The novel SARS-CoV-2 is a highly contagious virus that began in December of 2019 in Wuhan, China. Over 2 million people have been infected with the virus and about 150,000 deaths were reported in a time span of about 4 months since its inception (Li et al. 2020). An exponential increase in COVID-19 infections in the following 2 months after April to June of 2020 caused cases to reach more than 7 million and the death toll to reach more than 400,000 globally (Sanyaolu et al. 2021). The transition from a local epidemic into a worldwide pandemic was quick and profound.

In this review, a brief and general overview of SARS-CoV-2, which causes COVID-19, is summarized. A better understanding and perspective of COVID-19 is essential for efforts in reducing infection rates or “flattening the curve” in Layman’s term. Efforts and preventive measures need to be taken to reduce the negative impacts of COVID-19.

Structure, Attachment and Genome

The size of a coronavirus was determined by transmission electron microscopy to be around 60 to 140 nm in diameter (Zhu et al. 2020). The coronavirus is circular in shape and possesses an envelope (Mousavizadeh and Ghasemi 2021). The genome of the virus is also surrounded by a nucleocapsid. Four structural proteins are embedded throughout the SARS-CoV-2 virus. These include the spike (S) protein, the membrane (M) protein, the envelope (E) protein, and the nucleocapsid (N) protein (Boopathi et al. 2021). The N and M proteins are generally supportive for the virus: the N proteins protect the virus’s genetic material while the M and E proteins assist with assembly. The E and S proteins both assist in host cell receptor interactions, where the S proteins are the primary viral antigens. The S protein, or glycoprotein spike, is arguably the most important structural protein of the virus. It is a trimer, meaning three proteins, divided into the S1 head and the S2 stalk. These subunits play an important role in cell attachment and entry into the host cell either through fusion, where viral proteins are left on the plasma membrane of the host cell, or by endocytosis where the entire virus is engulfed by the invagination of the plasma membrane (Mousavizadeh and Ghasemi 2021). The S protein spike also facilitates the fusing of the phospholipid bilayer between the envelope of the virus and the

plasma membrane of the host allowing viral deposits into the cytosol. Host proteases, like furin and trypsin, assist in these conformational changes. Structural changes to the S protein are problematic in areas of pandemic control and medical intervention. Mutations and amino acid changes to the S protein are of clinical concern because they can cause antivirals and/or antibodies that were effective in previous strains to be ineffective (Abdullahi et al. 2020). These changes, however, are evolutionarily favorable for coronaviruses as it confers improved fitness and infection. Various COVID-19 strains have been identified since its inception in humans which include, as of December 10, 2021, the: alpha, beta, gamma, delta, and the recent novel omicron.

It is widely supported that the host angiotensin-converting enzyme 2 (ACE-2) receptor is the primary target for the COVID-19 virus (Mackenzie and Smith 2020; Mousavizadeh and Ghasemi 2021; Boopathi et al. 2021; Fehr and Perlman 2015). In addition to ACE-2, CD209 has also found to be a compatible receptor for the virus (Beniac et al. 2006). Supportive entry into the host cell is facilitated by the co-receptor transmembrane serine protease 2 (TMPRSS2) which is also found on the cellular surface of the host (Mollica et al. 2020). The novel COVID-19 virus has a tropism or affinity toward cells that express ACE-2 where higher expression of ACE-2 correlates to higher likelihood of infection (Lin et al. 2020). The COVID-19 virus is commonly associated with respiratory illness and infection where ACE-2 receptors are commonly found. However other systemic regions of the body also express ACE-2 receptors including: digestive, circulatory, urinary, endocrine, and integumentary systems (Li et al. 2020). Restricting the perspective of COVID-19 pathogenesis to just the respiratory track is limiting. Other locations of the body express varying levels of ACE-2 where the bloodstream is a source of mobility for the virus to infect other human tissues.

The genome of the coronavirus is a (+) single stranded RNA (ssRNA) where it ranges about 30 kb in size and possess at least six open reading frames (ORFs) (Chen et al. 2020). The (+) ssRNA means that the nucleic acid of the coronavirus is like the modified mRNA in eukaryotes meaning the strand is ready for translation. ORFs are the genetic sequences that the ribosomes read from. Approximately two thirds of the genome represented by ORF1a and ORF1b, or pp1a and pp1b, encode nonstructural proteins. The remaining genome towards the 3' end encodes important structural proteins like the S, M, N, and E. ORF1a and ORF1b overlap each other. This is common in viral genomes where a frameshift may occur due to ribosomal interactions with pseudoknots, also known as stem loops (Fehr and Perlman 2015). The occurrence of frameshift in the genomic translation is not entirely understood. However, it is a necessary sequence of events for protein translation, which can be an area of medicinal intervention. Like the modified finished mRNA in humans, the (+) ssRNA of the virus has a 5' cap and a 3' poly-A-tail. In humans, a segment of transcribed mRNA encodes one protein. This is not the case in viruses, in which an mRNA segment can encode multiple proteins that are later separated. The viral genome contains everything it needs for assembly, packaging, and release. The single viral genome encodes non-structural proteins (nsps), structural proteins, and accessory proteins followed by separation with proteases, enzymes that can cleave polyproteins.

Replication and Release

SARS-CoV-2 entry, as a result of ACE-2 binding and conformational changes, and release of its genomic material into the host cytoplasm is dependent on pH levels (Shang et al. 2021). Both the subunits of the S protein binding at the surface, and membrane fusion with the

endosome, the vesicle carrying the virus in the cytoplasm, requires acidification at low pH. Once the (+) ssRNA genome is released into the cytoplasm and the nucleocapsid has been degraded by proteases, replication can begin (Boopathi et al. 2021). The genome of coronavirus acts like the finished modified mRNA in humans meaning it is ready for translation by host ribosomes. The nsps translated from ORF1a and ORF1b form replication transcription complexes (RTC) where progeny RNA viruses will be produced. Double membrane vesicles (DMV) house the RTC (Chen et al. 2020). DMV are a signature feature of (+) RNA viruses where it manipulates the intracellular membranes into a machinery for viral synthesis (Wolff et al. 2020). It is suggested that the DMV offer protection to virus replication, in addition to viral synthesis, by evading immune cell detection, although its function is not entirely clear. RNA to RNA replication is processed through a viral enzyme known as RNA-dependent-RNA polymerase (RdRp), which is produced from the nsp 12 sequence reading frame (Gao et al. 2020). From the parent (+) ssRNA, RdRp will produce a (-) subgenomic RNAs (sgRNAs) which will then act as a template for producing the next progeny of (+) ssRNA viruses to be packaged. Each replication step produces an exponentially higher number than before allowing multiple viruses to be produced from a limited few. Synthesized structural proteins, including S, E and M, are embedded within the membranes of the endoplasmic reticulum (ER). The (+) ssRNA with the N protein, together constituting the nucleocapsid, buds into the ER membrane surfaced with structural proteins and gaining an envelope. The endoplasmic reticulum-Golgi intermediate compartment (ERGIC) will then facilitate virion release by exocytosis out of the host cell where it can infect new cells. This replication cycle then repeats.

Transmission

Viruses can be transmitted via various methods. These routes of entry and or exit include the respiratory tract, gastrointestinal (GI) tract, genital tract, fecal-oral tract, or the skin tract (Louten 2016). Viruses can also be transmitted from mother to child in vertical transmission. Generally, horizontal transmission is most common where viruses are passed from person to person. Viruses interact with epithelial cells and tissues lined with mucosa which is found widely in the human body. The novel SARS-CoV-2 is known to infect the respiratory tract where the surface is lined with mucosal epithelium. This respiratory virus is commonly spread directly by healthy individuals inhaling infected droplet particles (Mackenzie and Smith 2020). SARS-CoV-2 can also be spread indirectly via contact with fomites on contaminated surfaces. In addition to normal human respiration: sneezing and coughing contribute to a large release of these infected droplet particles. Whether the coronavirus is transmitted mainly through aerosols or droplets is unclear, but both are possibilities. Droplets are classified as large particles containing infectious virions whereas aerosols are characterized as smaller infectious droplet nuclei that is suspended within the air. Larger size droplets, >100 μm , are subjected to gravity and to fall within close range of release whereas smaller bioaerosols, <100 μm , travel longer distances and remain suspended in the environment (Jayaweera et al. 2020). Size differences contribute to infectivity. Droplets and aerosols can travel distances up to approximately 1, 3, and 6 meter from exhaling, coughing, and sneezing respectively where larger particles are heavily concentrated in proximity to the origin. Environmental factors, like temperature and ventilation, affect particle spread and infectivity by impacting evaporation of small aerosols and descent of large droplets. Therefore, in the occurrence of normal human respiration, a distance of about 2 meters, in concurrence with CDC guidelines, is recommended between individuals. In indoor settings, the 6 feet rule may

need to be adjusted due to the continuing circulation of viral particles. There was only a minor difference in SARS-CoV-2 infection rates between schools that implement a 3 feet distance rule and those who implement a 6 feet distance rule (Berg et al. 2021). The 2 meter, or 6 feet, distance is inconclusive as studies have shown viral particles can travel up to 10 meters (Setti et al. 2020). Wearing a face mask is a necessary intervention to prevent the spread of SARS-CoV-2, especially in indoor settings with less ventilation, which can increase viral particle travel distances in the air. Face masks should be considered in individuals without symptoms of SARS-CoV-2 as infections may be asymptomatic. As much as 59% of transmitted SARS-CoV-2 infections are from asymptomatic individuals contributing to the widespread pandemic (Johansson et al. 2021). Front-line healthcare workers are especially prone to contracting the virus due to close distance and exposure to patients infected with SARS-CoV-2 (Nguyen et al. 2020). Therefore, adequate and properly worn personal protective equipment (PPE) is essential for healthcare workers.

Pathogenesis and Virulence

The SARS-CoV-2 virus primarily infects the cells within the respiratory system where ACE-2 receptors are commonly found (Fehr and Perlman 2015). The nasal cavity or the nasopharynx area is highly susceptible to the onset of infection given infective droplets and aerosolized transmissions are inhaled through the nose. The time of COVID-19 incubation and disease development is around 2 to 14 days (Sanyaolu et al. 2021). This initial onset stage is usually asymptomatic, but the individual is considered infectious as the virus can be spread to others (Mason 2020). Individuals without symptoms contribute to superspreading events, where multiple people not wearing face masks contract the virus. Progression of the virus down the respiratory tract at the trachea marks the 2nd stage where inflammatory makers like cytokines can be detected. At this stage, symptoms are generally mild and flu like. Unresolved coronavirus infections by the immune system and proliferation within the lungs, containing alveolar tissues, marks the 3rd stage of the disease. At this stage, advanced medical treatment is required for pulmonary symptoms like pneumonia where the air sacs are filled with fluid. ACE-2 functions as a regulatory protein in blood vessel dilations (Kuba et al. 2013). Excessive vasodilatation is harmful for healthy organs as tissues swell and fluids accumulate. SARS-CoV-2 downregulates the ACE-2 activity, thus promoting vasodilatation and fluid retention as in the case of pneumonia. SARS-CoV-2 is not restricted to the respiratory system. Other systemic regions of the body also express the ACE-2 receptor and are therefore viable hosts for the virus (Li et al. 2020). The alveoli are surrounded by capillaries where virions can enter into the bloodstream and relocate to other parts of the body. The epithelial lining of the GI system, the circulatory system, and the integumentary system are also suitable for the virus inducing symptoms including diarrhea, vessel inflammation, and rashes respectively (Larsen et al. 2020). Heart cells also express ACE-2 receptors, rendering them prone to infection (Li et al. 2020). Although myocarditis, known as heart inflammation, is uncommon, individuals infected with COVID-19 have a higher risk of developing this condition (Boehmer et al. 2021). Individuals with cardiovascular, digestive, or other medical complications are at a higher risk of co-morbidities when infected with SARS-CoV-2 (Mousavizadeh and Ghasemi 2021). Age and sex are factors that affect the degree of SARS-CoV-2 infection and severity, where older individuals have higher risk. More specifically, men have a higher risk of infection and severity than women (Pijls

et al. 2021). Being over 70 years of age is also a risk factor because body ailments increase and immune effectiveness decrease. Men over the age of 70 are at higher risk of needing medical care for SARS-CoV-2 infection than women.

Treatment and Intervention

Viral entry and replication mechanisms are great targets for developing novel drug therapies since these processes are unique to viruses. Different approaches are available in combating SARS-CoV-2, but interventions remain mostly traditional as urgency prompts the reuse of old drugs while simultaneously researching new treatments (Boopathi et al. 2021). As such, combining anti-virals and immunomodulators, drugs that enhance the immune system, is a great method to combat the COVID-19 disease (Feuillet et al. 2021). The majority of asymptomatic infections can be attributed to the effectiveness of individuals' immune systems that clear the virus before symptoms arise. Cell production of interferons are natural defenses against viral pathogens support the immune response. Anti-viral drugs combined with cell production of interferons can reduce viral complications early. Prolonged inflammatory signaling can be dangerous as cytokine storms can result in putting the individual in acute respiratory distress. Other options range from drug treatments to vaccine delivery. Remdesivir and paxlovid are examples of antivirals. Anti-parasitic drugs can also be used to target viral infections: examples include chloroquine and hydroxychloroquine. Lastly, monoclonal antibodies and vaccines will also be discussed as methods of intervention.

Remdesivir is an analog drug that interferes with COVID-19 genome synthesis (Beigel 2021). The remdesivir drug will attach itself onto the RdRp, inhibiting viral replication since the enzyme is needed for producing progeny viruses. The drug reduces respiratory symptoms due to inhibited viral activity and improves patient outcomes. Despite conflicting results of remdesivir usage, remdesivir does benefit overall.

Paxlovid, also known as ritonavir, also interferes with enzymes, specifically targeting viral proteases. Proteases play a function in cleaving viral polyproteins and assist in replication by replicase enzyme, like RdRp, maturation (Gioia et al. 2020). Paxlovid inhibits and blocks the function of protease. As a result, the virion cannot perform genomic replication. Pfizer is the lead pharmaceutical company in this oral anti-viral drug.

Hydroxychloroquine and Chloroquine are drugs to treat parasitic infections, like malaria, and auto-immune diseases, like lupus and rheumatoid arthritis (Boopathi et al. 2021). Its usage in SARS-CoV-2 treatment interferes with low pH that would normally be needed for viral entry and genomic release into the cytoplasm (Shang et al. 2021). Since acidification of the vesicle containing the virion is required for genomic release, hydroxychloroquine and chloroquine increases pH concentration resulting in a more basic environment. This interferes with the COVID-19 replication and reduces symptoms like pneumonia in patients. The usage of these anti-parasitic drugs in fighting against COVID-19 is controversial as there have been mixed results in patient health and side effects.

Monoclonal antibodies (mAbs) work by interfering with compatible antigen-to-receptor match. This is like the concept of a lock and a key analogy discussed in the introduction. In this analogy, the S protein spike of SARS-CoV-2 is the key and the ACE-2 receptor on host cells are the lock. Monoclonal antibodies act as a blocker and attach to the epitope, the S protein spike, of the virus. Therefore, the S protein key cannot interact with the ACE-2 receptor lock. Different types of mAbs include casirivimab, imdevimab, bamlanivimab, and etesevimab (Pinna et al.

2021). Convalescent plasma works in a similar manner. The antibodies are retrieved from COVID-19 survivors that have produced natural antibodies against the virus. These antibodies can be donated and administered to COVID-19 patients intravenously (IV), especially those with immuno-compromised conditions, to assist in recovery. Like with many therapies, patient outcomes vary.

Vaccines are a subject of strong controversy and debate among the general population. Media literacy and scrutiny is necessary in a world of technology where information is easily obtained from different sources. False information, rumors, and facts can be readily shared alongside scientific evidence. Accurately accessing and learning information online can lessen the media hysteria on the topic of vaccination. The purpose of the vaccine is to prepare the body with a false infection to invoke immunity towards the actual disease (Ndwandwe and Wiysonge 2021). Vaccine development is a long and arduous process with frequent testing and clinical trials that range around 15 years until distribution to the public. The SARS-CoV-2 vaccine was developed in a matter of 1 year, leading to hesitancy in individuals not choosing to get immediate vaccination, due to the short time development. However, vaccinated individuals are not fully protected and are still able to receive and transmit the SARS-CoV-2 disease (Brown et al. 2021). Among the various kinds of vaccines, common vaccines include: attenuated, inactivated, toxoid, subunit, and vector (Clem 2011). An attenuated vaccine uses a live weakened version of the virus to stimulate an immune response. Inactivated vaccines use a dead version of the virus instead. Toxoid and subunit are similar because parts and fragments of the virus, like the S spike protein for SARS-CoV-2, is used to stimulate an immune response. Lastly, vector vaccines, also known as genomic vaccine, uses microorganisms to deliver a genomic sequence to the cell to express. Expression of viral proteins can then induce an immune response. For the SARS-CoV-2 mRNA vaccine, pharmaceutical companies like Pfizer, Moderna, and Johnson & Johnson use this method with modifications from each other.

Discussions

A brief overview of viruses was presented followed by detailed SARS-CoV-2 virology. This review is limited to the novel SARS-CoV-2 whereas a related coronavirus pathogen in the Middle East known as MERS-CoV causes similar havoc. The novel SARS-CoV-2 took the world by storm. The quick transition from a local endemic situation in Wuhan, China to worldwide pandemic showcases the capabilities of viruses. Viruses are extremely small non-living microorganisms that have the potential to cause large obstacles in health care, world trade, and economic flow. The anatomy of a virus is mainly structural proteins and nucleic acids. These general features make viruses extraordinary simple particles but mechanistically complex at the same time. Viruses are programmed to do what they do by cycles of infections and replications. As Van and Mahy (2004) state, viruses live a borrowed life. Viruses cannot be eradicated. Instead, finding ways to manipulate the viral mechanisms and utilize them for beneficial causes is ideal. This approach is already in development as viral vectors are used in medical intervention, and with the mRNA vaccine to combat the novel SARS-CoV-2. Other forms of vector treatment in the foreseeable future are promising innovations in the field of medicine. The infectivity of the SARS-CoV-2 pandemic is facilitated by human actions. In the era of international trade and commerce, humans are the vehicles by which viruses enter multiple regions. Given the current circumstances with the COVID-19 pandemic, it is appropriate that this

review serves as a source of information to help readers understand viruses and to appreciate their complexity.

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